Home Dampness and Respiratory Morbidity in Children[1-4]

BERT BRUNEKREEF,[5] DOUGLAS W. DOCKERY,[6] FRANK E. SPEIZER, JAMES H. WARE, JOHN D. SPENGLER, and BENJAMIN G. FERRIS

SUMMARY This study examined the relationship between measures of home dampness and respiratory illness and symptoms in a cohort of 4,825 eight- to 12-yr-old children living in six U.S. cities. Home dampness was characterized from questionnaire reports of mold or mildew inside the home, water damage to the home, and the occurrence of water on the basement floor. Symptoms of respiratory and other illness were collected by questionnaire. Pulmonary function was measured by spirometry. Signs of home dampness were reported in a large proportion of the homes. In five of the six cities, one or more of the dampness indicators were reported in more than 50% of the homes. The association between measures of home dampness and both respiratory symptoms and other nonchest illness was both strong and consistent. Odds ratios for molds varied from 1.27 to 2.12, and for dampness from 1.23 to 2.16 after adjustment for maternal smoking, age, gender, city of residence, and parental education. The relationship between home dampness and pulmonary function was weak, with an estimated mean reduction of 1.0% in FEF_{25-75} associated with dampness and 1.8% with molds. We conclude that dampness in the home is common in many areas of the United States and that home dampness is a strong predictor of symptoms of respiratory and other illness symptoms among 8- to 12-yr-old children. AM REV RESPIR DIS 1989; 140:1363-1367

Introduction

Dampness in the home is a potential risk factor for respiratory illness in part through the action of (micro)organisms that thrive in damp environments. Dust mites (Pyroglyphidae) are a well-known source of allergenic substances (1-5). They thrive at relative humidities greater than 70%, and they are found in mattresses, carpets, and dust on surfaces moist enough to support them. Dust mites are most abundant at the end of the summer, after a prolonged period of high indoor humidity (6-10). They can survive low humidity conditions to a certain extent, but their numbers are reduced significantly in homes that are very dry in winter (10). The prevalence of mites and allergens in house dust decreases with increasing altitude (1, 11, 12), presumably because wintertime humidity is lower at higher altitudes. Few mites are found in dusts obtained in dry climates (13, 14). Murray and coworkers (15) reported a 10- to 20-fold increase in sensitization to mite extracts in children living in a damp climate when compared with children living in a dry climate. Sensitization to cat and dog dander was comparable to the two groups. Long before the house dust mite was shown to be responsible for allergy to house dust (1), it was known that asthmatics normally living at low altitudes suffered far fewer attacks at high altitudes, and that asthmatic patients generally had negative skin tests for dusts collected at high altitudes (16). Recently, an increased prevalence of asthma in Papua, New Guinea, has been associated with the increased use of blankets containing large numbers of dust mites (17).

Molds are another source of respiratory allergens (18-21). Mold species have critical relative humidities ranging from less than 80% to more than 90% (18). Some genera have stronger allergenic properties than do others, and within a genus, there can be considerable variation in allergenic potential among species (21). Mold growth in homes can cause severe respiratory disease requiring hospitalization (22-24). Typical causes of abundant mold growth include leaks in the roofs or walls, urinating pets, improper carpet cleaning, leaky plant pots, and the use of a cold mist vaporizer (22-24). Molds are ubiquitous in ambient air, and in dry homes, the presence of molds appears mainly to be related to their presence in outdoor air (25). *Penicillium* and *Aspergillus* are among the molds typically found in residences (26-31). Within the allergic population, the prevalence of mold allergy has been estimated to be 2 to 30% (32).

Despite this information about the potentially harmful effects of home dampness on respiratory health, relatively few epidemiologic studies have investigated the health effects of dampness. A study from the United Kingdom reported an association in a group of about 200 children between the prevalence of respiratory symptoms and relative humidity in their bedrooms (33). Lesourd and coworkers (34) reported a trend toward increasing prevalence of cutaneous delayed-type hypersensitivity to a battery of ubiquitous antigens in white, Hispanic, and black schoolchildren. These investigators attributed this trend to the increased risk of exposure to microorganisms in poorer homes. Varekamp and Voorhorst (35) and Leupen (36) reported that patients with bronchial asthma were more likely to live in damp homes than were control subjects.

This report utilizes information on home characteristics and respiratory health of children participating in a large, ongoing epidemiologic study of air pollution

(*Received in original form October 25, 1988 and in revised form April 17, 1989*)

[1] From the Respiratory Epidemiology Program, Department of Environmental Science and Physiology, Harvard School of Public Health, and Channing Laboratory, Department of Medicine, Harvard Medical School, Brigham and Women's Hospital, Boston, Massachusetts.

[2] Supported in part by Grants ES-00002 and ES-01108 from the National Institute of Environmental Health Sciences, by Cooperative Agreement CR811650 from the Environmental Protection Agency, and by Contract RP-1001 from the Electric Power Research Institute.

[3] This report has not been subjected to the Environmental Protection Agency's required peer and policy review and therefore does not necessarily reflect the reviews of the agency, and no official endorsement should be inferred.

[4] Correspondence and requests for reprints should be addressed to Dr. Douglas W. Dockery, Respiratory Epidemiology Program, Harvard School of Public Health, 665 Huntington Ave., Boston, MA 02115.

[5] Recipient of a grant from the Netherlands Organization for the Advancement of Pure Research (ZWO).

[6] Recipient of a Faculty Development Award from the Mellon Foundation.

and respiratory health to explore the relationship between moisture in the home and respiratory symptoms in children.

Methods

Study Population

The study population consists of 6,273 schoolchildren living in six U.S. communities originally selected for their historic outdoor air pollution levels. These communities include Watertown, MA; Kingston and Harriman, TN; a geographically defined area in the southeast corner of St. Louis, MO; Steubenville and Mingo Junction, OH; Portage, Pardeeville, and Wyocena, WI, and a random sample of 50% of the schools in Topeka, KS.

Initially, approximately 1,000 children were enrolled in each city between 1983 (Watertown) and 1986 (Topeka). The cohort was drawn from the second through the fifth grades, with the number of grades depending on the size of the school population. Overall, 95.9% of the invited children participated. Sample sizes in participating communities varied between 832 (Watertown) and 1,135 (St. Louis). One year after the first examination, the children were invited to participate in a follow-up study. A total of 5,395 children participated, ranging from 735 in Watertown to 1,005 in Topeka. For 5,321 of these participants, both a questionnaire and a pulmonary function test were obtained. This report is based on the 4,625 white children who were 7 to 11 yr of age at the start of the study and who were seen again at the 1-yr follow-up examination.

Health, Exposure, and Demographic Information

At both examinations, a questionnaire was given to each child to be completed by a parent or guardian and returned to school. Information regarding respiratory illnesses and symptoms was requested in a format equivalent to that recommended by the Epidemiology Standardization Project (37). Of the respiratory illnesses and symptoms, responses concerning doctor-diagnosed respiratory illness before 2 yr of age, bronchitis in the previous year, persistent cough (for 3 months of the year or more), persistent wheeze (most days or nights or apart from colds), chest illness that kept the child at home for 3 days or more, and an index of lower respiratory illness (bronchitis, cough, or chest illness) were considered. The questionnaire-based definition of these symptoms has been reported previously (38). In addition, the occurrence of doctor-diagnosed asthma in the past year has been considered, as home dampness appears to increase the presence of substances that cause and/or aggravate asthma. The occurrence of hay fever in the past year has been considered, as this may be indicative of the child's sensitivity to respiratory allergens. Other nonchest illnesses that restricted the child's activities for 3 days or more also were investigated, as molds may release biologically active substances that lead to systemic effects (39). These other nonchest illnesses were defined by the question: "In the past year, has this child had any other major illness or accident that restricted his/her activities for a week or more?" This immediately followed the analogous question on chest illnesses.

The effect of home dampness on respiratory symptoms was evaluated separately for children with asthma or asthmatic symptoms. Persistent wheeze is often considered to be a marker for asthma in children. In this sample, more than half of the children with persistent wheeze did not report doctor-diagnosed asthma. Three groups of children were considered: children with doctor-diagnosed asthma, children with persistent wheeze but without doctor-diagnosed asthma, and children with neither asthma nor persistent wheeze.

In the initial as well as the follow-up questionnaire, information was asked about the family's smoking habits. Exposure to environmental tobacco smoke was expressed as the presence or absence of a mother who smokes in the home, a variable previously shown to affect childhood respiratory illness rates (38). Paternal smoking is highly correlated with maternal smoking, and it has been shown to affect childhood respiratory illness rates, although not as strongly as maternal smoking (38). Only maternal smoking was controlled in this analysis. The mean number of years of schooling of the parents (< 9, 9–12, > 12) was used as a proxy for socioeconomic factors that might influence respiratory health or symptom reporting.

The children were examined at school, where their weight and height in stockinged feet were measured. Forced expiratory maneuvers were performed on a recording spirometer (Survey Spirometer; Warren E. Collins, Braintree, MA) in a sitting position with free mobility without a noseclip. A detailed description of the measurement procedure has been given elsewhere (40).

In the follow-up questionnaire, three questions were included regarding potential moisture problems in the home: (*1*) Does water ever collect on the basement floor? (*2*) Has there ever been water damage to the building? (*3*) Has there ever been mold or mildew on any surface inside the home? From these, a fourth variable, home dampness, was created (dampness absent if answers to questions 1 to 3 were negative, present if any were positive).

Indoor air pollution measurements, including humidity, were made in a stratified random sample of the homes of about 1,800 children (41). Relative humidity of the indoor air is less important for the growth of mites and fungi than the dampness of specific surfaces or parts of the building structure. These measurements will be reported separately.

The relationship between the questionnaire indicators of dampness and the respiratory health outcomes is the subject of this report.

Statistical Methods

As a first step, symptom prevalences were calculated for each category of the home dampness variables. To investigate potential differences between cities, odds ratios were calculated for a number of relevant symptoms against the combined dampness variable for each city. In the next step, logistic regression models were constructed in which the association between home dampness variables and symptoms was adjusted for age, sex, parental education, maternal smoking, and city of residence.

Pulmonary function was measured by FEV_1, FVC, and FEF_{25-75}. The logarithm of each pulmonary function variable was regressed on an indicator of sex and on the logarithm of age, height, and weight plus maternal smoking, parental education, and indicator variables for the city of residence. Previously published analyses of pulmonary function of preadolescent children (40) have shown that this logarithmic transformation produces a linear relationship with constant variance. The residuals from these models were compared between categories of the home dampness variables by *t* test.

All statistical analyses were performed using the SAS Statistical Analysis System (42) on a Compaq 286 Deskpro personal computer.

Results

Reporting rates for the different home dampness variables are given for each city in table 1. Molds and mildew were reported in almost 40% of homes in Kings-

TABLE 1

REPORTING RATES FOR HOME DAMPNESS VARIABLES IN SIX U.S. CITIES

City	Reporting Rate for: Molds (%)	Water Damage (%)	Basement Water* (%)	Dampness† (%)
Kingston, TN	38.1	12.1	11.3	45.7
Steubenville, OH	27.9	14.7	38.0	55.6
Watertown, MA	20.9	16.4	42.0	55.8
St. Louis, MO	26.9	23.0	39.4	56.4
Topeka, KS	33.0	22.2	30.0	56.9
Portage, WI	35.4	16.0	33.3	58.2

* Includes homes with no basements.

† Dampness is defined as molds, water damage, or water in basement.

TABLE 2

SYMPTOM PREVALENCE FOR EACH OF THE HOME DAMPNESS VARIABLE CATEGORIES

	Dampness Variable							
	Molds		Water Damage		Basement Water		Dampness	
Symptom	Yes	No	Yes	No	Yes	No	Yes	No
Wheeze	14.8	8.9	15.0	9.9	11.2	10.4	12.2	9.0
Cough	11.7	6.1	10.9	7.2	9.6	7.1	10.1	5.1
Bronchitis	11.1	7.0	10.3	7.8	7.9	8.5	9.0	7.4
Chest illness	10.7	7.7	11.4	8.0	10.0	7.9	9.9	6.8
Lower respiratory illness	24.5	16.2	23.8	17.7	20.8	17.9	22.0	14.8
Respiratory illness before age 2	12.3	8.4	10.8	9.3	10.0	9.4	10.8	8.1
Asthma	5.4	4.4	5.4	4.5	5.2	4.5	5.1	4.1
Hay fever	23.2	15.8	22.2	17.4	19.4	17.6	19.8	16.3
Nonchest illness	12.5	9.1	13.7	9.5	11.7	9.6	11.9	8.3

ton/Harriman, TN. Reporting rates in other communities ranged from 20.9% in Watertown to 35.4% in Portage. A high rate of water damage was found in St. Louis, MO (23.0%), and water was reported to collect occasionally in the basement in approximately 40% of the homes in Watertown, MA, St. Louis, MO, and Steubenville, OH. The low reporting rate in Kingston/Harriman, TN is partly related to the fact that few homes in the sample had basements (63.3% compared with 78.4% in Topeka and 93.7% to 98.3% in the other communities).

Children living in homes with indications of dampness had consistently higher rates of respiratory symptoms than did children living in homes without these indications (table 2). City-specific unadjusted odds ratios for the association between respiratory symptoms and dampness were remarkably consistent across the six communities (table 3). Similar patterns were found with the other measures of home dampness (results not shown). These city-specific values demonstrate a strong and geographically consistent association between dampness and most childhood respiratory symptoms.

When the data were combined across cities and adjusted for other predictors of respiratory symptoms, respiratory illness and symptoms, including asthma and hay fever and other nonchest illnesses, had associations with molds varying from 1.27 to 2.12 and with dampness varying from 1.23 to 2.16 (table 4). All but one of these associations were statistically significant, although the association was weakest for asthma. Similar results were obtained for water damage and water in the basement.

To assess the effects of asthma and wheeze on the association between home dampness and other respiratory symptoms, the population was stratified into three groups: doctor-diagnosed asthmatics, wheeze without doctor-diagnosed asthma, and children with neither wheeze nor asthma. The results for molds, expressed as prevalence ratios, are given in table 5. Prevalence ratios were used for this comparison because wide variations in prevalence in the unexposed groups made odds ratios difficult to interpret. Ratios were consistently smallest among asthmatics, but this is explained in part by the higher reporting rates among children not exposed to dampness.

After adjusting for age, height, weight, sex, city of residence, parental education, and maternal smoking, there was no difference in level of FEV_1 or FVC with the presence of home dampness (table 6). There was some indication that FEF_{25-75} was negatively related to molds.

It could be argued that the excess of persistent wheeze among children living in damp homes was due to overreporting of symptoms by parents living in those homes. If such overreporting did occur, the group of children living in damp homes and reported to be symptomatic would be a healthier group than the symptomatic children living in dry homes. To investigate this issue, we investigated the relationship between persistent wheeze and pulmonary function separately for children living in homes with and without reported molds. Persistent wheeze was associated with an 8.5% deficit in FEF_{25-75} (95% CI, 5.6% to 11.2%) among children in homes with no molds, and a 9.2% deficit (95% CI, 5.5% to 12.7%) in homes with molds af-

TABLE 3

CITY-SPECIFIC ODDS RATIOS FOR EFFECT OF DAMPNESS ON SELECTED RESPIRATORY ILLNESS SYMPTOMS IN SIX U.S. CITIES

	Respiratory Illness Symptom				
City	Wheeze	Cough	Bronchitis	Chest Illness	Respiratory Illness before Age 2
Kingston, TN	1.51	3.72*	1.66*	1.31	1.55*
Steubenville, OH	1.48	1.42	1.40	1.78*	1.42
Watertown, MA	1.57	1.74	1.77	1.57	1.72
St. Louis, MO	1.53	1.98*	1.19	1.56	0.94
Topeka, KS	1.40	2.48*	1.09	1.86*	1.77*
Portage, WI	1.23	2.00*	0.90	1.38	1.43

* $p < 0.05$

TABLE 4

ASSOCIATIONS BETWEEN HOME DAMPNESS AND SYMPTOMS OF CHILDHOOD RESPIRATORY AND OTHER ILLNESS, ADJUSTED FOR AGE, SEX, CITY OF RESIDENCE, PARENTAL EDUCATION, AND MATERNAL SMOKING, IN SIX U.S. CITIES

	Estimated Odds Ratios for	
Symptom	Molds	Dampness
Wheeze	1.79 (1.44, 2.32)*	1.23 (1.10, 1.39)
Cough	2.12 (1.64, 2.73)	2.16 (1.64, 2.84)
Bronchitis	1.48 (1.17, 1.87)	1.32 (1.05, 1.67)
Chest illness	1.40 (1.11, 1.78)	1.52 (1.20, 1.93)
Lower respiratory illness	1.57 (1.31, 1.87)	1.68 (1.41, 2.01)
Respiratory illness before age 2	1.42 (1.12, 1.80)	1.40 (1.11, 1.78)
Asthma	1.27 (0.93, 1.74)	1.42 (1.04, 1.94)
Hay fever	1.57 (1.31, 1.87)	1.26 (1.06, 1.50)
Nonchest illness	1.40 (1.13, 1.74)	1.55 (1.25, 1.93)

* 95% confidence limits in parentheses.

TABLE 5

ASSOCIATIONS BETWEEN REPORTED MOLDS IN THE HOME AND RESPIRATORY SYMPTOMS IN ASTHMATICS, NONASTHMATIC WHEEZERS, AND NONASTHMATIC NONWHEEZERS IN SIX U.S. CITIES

	Estimated Prevalence Ratios for:		
Symptom	Asthmatics (n = 214)	Nonasthmatic Wheezers (n = 297)	Nonasthmatic Nonwheezers (n = 3,799)
Cough	1.50*	1.73*	1.59*
Bronchitis	0.66	1.41	1.74*
Chest illness	1.20	1.46	1.13
Lower respiratory illness	1.20	1.37*	1.39*
Respiratory illness before age 2	1.07	1.13	1.31*
Hay fever	1.04	1.46*	1.38*
Nonchest illness	0.99	1.51	1.31*

* $p < 0.05$

TABLE 6

ASSOCIATIONS BETWEEN HOME DAMPNESS VARIABLES AND PULMONARY FUNCTION IN CHILDREN, ADJUSTED FOR AGE, HEIGHT, WEIGHT, GENDER, CITY OF RESIDENCE, PARENTAL EDUCATION, AND MATERNAL SMOKING, IN SIX U.S. CITIES (n = 3,655)

Home Dampness Variable	Pulmonary Function Variable	Percent Difference Associated with Home Dampness*
Molds	FVC	0.44 (−0.27, 1.15)†
	FEV_1	0.03 (−0.75, 0.82)
	FEF_{25-75}	−1.62 (−3.19, −0.02)
Water damage	FVC	0.25 (−0.61, 1.12)
	FEV_1	0.35 (−0.59, 1.30)
	FEF_{25-75}	0.46 (−1.49, 2.45)
Basement water	FVC	0.16 (−0.54, 0.87)
	FEV_1	−0.14 (−0.92, 0.65)
	FEF_{25-75}	−1.14 (−2.74, 0.44)
Dampness	FVC	−0.09 (−0.75, 0.58)
	FEV_1	−0.21 (−0.93, 0.52)
	FEF_{25-75}	−1.06 (−2.55, 0.44)

* Difference in mean pulmonary function, expressed as percentage of the grand mean, between children living in damp homes and children living in dry homes.
† 95% confidence interval.

ter adjusting for age, sex, height, city of residence, parental education, and maternal smoking. FEV_1 was similarly reduced in children with persistent wheeze, irrespective of exposure to molds. Thus, children reported to have persistent wheeze had similar pulmonary function deficits whether they lived in dry or in damp homes, a result not consistent with the hypothesis of overreporting.

The questionnaire data were used to investigate the association between the home dampness variables and a number of potential determinants of home dampness: the use of humidifiers or dehumidifiers, heating system, type of building, and age of building. There were more dehumidifiers in use in homes where molds and mildew were reported (42.0 versus 28.7%). Molds and mildew were reported in 34.9% of detached single family homes (which constituted 76% of the total sample), and in 17.3% of two-family homes (with 13% the only other major category). Molds and mildew were reported in 34.8 to 43.4% of homes built between 1940 and 1969, and in 26.1 to 31.9% of homes built either before 1940 or after 1969. Water damage was reported in 22.0% of the homes built before 1930 and in 9.9% of homes built after 1979, with increasing reporting rates with increasing age of the homes in between. Water in the basement was reported in 47.8% of homes built before 1930, decreasing gradually to a reporting rate of 11.2% in homes built after 1979. No other associations emerged from this analysis.

Discussion

The results presented in this report suggest a consistent and strong association between reported dampness in the home and childhood respiratory symptoms. This association remained after adjustment for city of residence, maternal smoking, age, sex, and parental education.

One explanation for these findings could be that people with children experiencing respiratory symptoms report dampness in their homes more readily than those whose children are not symptomatic. This seems unlikely, however, because the potential role of home dampness as a risk factor for respiratory illness has not received the public attention accorded to other risk factors such as parental smoking and the use of unvented combustion appliances.

Case studies (7, 10, 23) have documented increased mite populations and mold growth in damp homes. These organisms have, however, been associated primarily with causation and/or worsening of asthma. Interestingly, we find that reported asthma is the only respiratory symptom *not* consistently associated with dampness in the home. The symptom "persistent wheeze," which is closely associated with reported asthma in the data, is associated with dampness in the home. Also, the relationships with dampness were stronger among nonasthmatic children with or without persistent wheeze than among the asthmatic children (table 5). It could be argued that parents of children with doctor-diagnosed asthma tend to move or modify the home environment to make it as healthy as possible for their children.

The respiratory symptoms used in the analysis were strongly correlated. A child with one symptom was four times to more than ten times as likely to have one or more of the other respiratory symptoms than a child not having the index symptom. This makes it difficult to separate the associations between dampness in the home and the various respiratory symptoms. It is also possible that a relatively high percentage of children reporting symptoms other than wheeze or asthma have reactive airway disease. Hallett and Jacobs (43) reported that reactive airway disease was present in 80% of patients presenting with acute bronchitis. Molds have been associated with respiratory symptoms and diseases other than wheeze and asthma (23, 24).

A recent study in Edinburgh, Scotland found a highly elevated prevalence of respiratory symptoms among children living in damp homes (44). The prevalence of wheeze in the past year was as high as 38.1% in homes in which molds were reported to be present in the child's bedroom compared with 10.5% in homes where no molds were reported. There was no clear relationship between home

dampness and bronchial lability, however, and this was interpreted as evidence that the association between home dampness and respiratory symptoms could be due to overreporting of symptoms (or a greater awareness of symptoms) among parents of children living in damp homes. The prevalence of wheeze was much higher in the damp homes in Edinburgh than in the damp homes in this study. In this study, the prevalence difference between homes with and without reported molds was only 6% (14.8 versus 8.9%). The mean of FEF_{25-75} measurements was also lower among children living in damp homes, although only the association with molds was statistically significant. We also found that children with persistent wheeze had very similar deficits in FEF_{25-75} and FEV_1 in dry and damp homes. Overreporting of wheeze in damp homes would have diluted the association between wheeze and pulmonary function level present.

Another interesting finding is the association between home dampness and reported illnesses other than those of the chest. Comparable associations have not been found in this population between smoking in the home and other nonchest illnesses (45). Nonchest illnesses may include various illnesses of the upper respiratory tract such as head colds, rhinitis, and sinusitis, which may be considered respiratory illnesses, and the association with dampness indicators is plausible. A recent study from the United Kingdom (46) has also suggested that home dampness is related to nonrespiratory symptoms.

These findings have implications both for further studies of indoor pollutants and for health policy. The effect of molds or dampness is comparable in size to the effect of passive smoking (44). Whether the respiratory illnesses produced by passive smoking and molds and dampness have similar long-term significance is unknown. Further investigation of childhood respiratory illnesses will require consideration of both of these variables simultaneously.

References

1. Voorhorst R, Spieksma-Boezeman MIA, Spieksma FThM. Is a mite (*Dermatophagoides* sp.) the producer of the house-dust allergen? Allerg Asthma 1964; 10:329–34.
2. Voorhorst R, Spieksma FThM, Varekamp H, Leupen MJ, Lyklema AW. The house dust mite (*Dermatophagoides pteronyssinus*) and the allergens it produces. Identity with the house dust allergen. J Allergy 1967; 39:325–39.
3. Smith JM. Clinical findings in children with allergy to the house dust mites. Acta Allerg 1970; 25:37–40.
4. Korsgaard J. Mite asthma and residency. Am Rev Respir Dis 1983; 128:231–5.
5. Andersen I, Korsgaard J. Asthma and the indoor environment: assessment of the health implications of high indoor air humidity. In: Berglund B, Lindvall T, Sundell J, eds. Indoor air. Vol. 1. Stockholm: Swedish Council for Building Research, 1984; 79–86.
6. Bronswijk JEMH van. Hausstaub-Oekosystem und Hausstaub-Allergen(e). Acta Allerg 1972; 27: 219–28.
7. Bronswijk JEMH van. *Dermatophagoides pteronyssinus* (Trouessart, 1897) in mattress and floordust in a temperate climate (Acari: Pyroglyphidae). J Med Entomol 1973; 10:63–70.
8. Murray AB, Zuk P. The seasonal variation in a population of house dust mites in a North American city. J Allergy Clin Immunol 1979; 64:266–9.
9. Arlian LG, Bernstein IL, Gallagher JS. The prevalence of house dust mites, *Dermatophagoides* spp, and associated environmental conditions in homes in Ohio. J Allergy Clin Immunol 1982; 69: 527–32.
10. Korsgaard J. House dust mites and absolute indoor humidity. Allergy 1983; 38:85–92.
11. Berrens L, Young E, Zuidema P. A comparative chemical and clinical investigation of house dust extracts from alpine and lowland regions. Acta Allerg 1971; 26:200–12.
12. Vervloet D, Penaud A, Razzouk H, *et al.* Altitude and house dust mites. J Allergy Clin Immunol 1982; 69:290–6.
13. Voorhorst R. Quantitative aspects of the problem of house dust atopy and house dust mites. Acta Allerg 1970; 25:237–54.
14. Sørensen H, Gravesen S, Lind P, Schwartz B, Ashoor AA, Maglad S. The occurrence of indoor allergens in Saudi Arabia. Ann Allergy 1985; 54: 530–3.
15. Murray AB, Ferguson AC, Morrison BJ. Sensitization to *Dermatophagoides farinae* (Df) and *D. pteronyssinus* (Dp) in different climatic areas (abstract). J Allergy Clin Immunol 1984; 73:158.
16. Storm van Leeuwen W, Varekamp H, Bien L. Asthma bronchiale und Klima. Klin Wochenschr 1924; 3:520–3.
17. Dowse GK, Turner KJ, Stewart GA, Alpers MP, Woolcock AJ. The association between *Dermatophagoides* mites and the increasing prevalence of asthma in village communities within the Papua New Guinea highland. J Allergy Clin Immunol 1985; 75:75–83.
18. Gravesen S. Fungi as a cause of allergic disease. Allergy 1979; 34:134–54.
19. Salvaggio J, Aukrust L. Mold induced asthma. J Allergy Clin Immunol 1981; 68:327–46.
20. Holmberg K. Moulds as an agent in respiratory symptoms. In: Berglund B, Lindvall T, Sundell J, eds. Indoor air. Vol 3. Stockholm: Swedish Council for Building Research, 1984:233–8.
21. Reed CE. What we do and do not know about mould allergy and asthma. J Allergy Clin Immunol 1985; 76:773–5.
22. Solomon WR. Fungus aerosols arising from cold mist vaporizers. J Allergy 1974; 54:222–8.
23. Kozak PP, Gallup J, Cummins LH, Gillman SA. Currently available methods for home mould surveys II. Examples of problem homes studied. Ann Allergy 1980; 45:167–75.
24. Fergusson RJ, Milne LJR, Crompton GK. Penicillium allergic alveolitis: faulty installation of central heating. Thorax 1984; 39:294–8.
25. Richards M. Atmospheric mould spores in and out of doors. J Allergy 1954; 25:429–39.
26. Gravesen S. Identification and quantitation of indoor airborne microfungi during 12 months from 44 Danish homes. Acta Allerg 1972; 27:337–54.
27. Hirsch SR, Sosman JA. A one-year survey of mould growth inside 12 homes. Ann Allergy 1976; 36:30–8.
28. Solomon WR. A volumetric study of winter fungi prevalence in the air of mid-western homes. J Allergy Clin Immunol 1976; 57:46–55.
29. Kozak PP, Gallup J, Cummins LH, Gillman SA. Factors of importance in determining the prevalence of indoor moulds. Ann Allergy 1979; 43: 88–94.
30. Calvo MA, Guarro J, Suarez G, Ramirez C. Airborne fungi in the air of Barcelona, Spain IV. Studies of the spore content of air in dwellings. Ann Allergy 1980; 44:228–34.
31. Beaumont F, Kauffman HF, Sluiter HJ, Vries K de. A volumetric-aerobiologic study of seasonal fungi prevalence inside and outside dwellings of asthmatic patients living in northeast Netherlands. Ann Allergy 1984; 53:486–92.
32. Gravesen S. Indoor airborne mould spores. Allergy 1985; 40(Suppl 3:21–3).
33. Melia RJW, Florey C du V, Morris RW, Goldstein B, John H, Clark C, Craighead I, Mackinlay J. Childhood respiratory illness and the home environment II. Association between respiratory illness and nitrogen dioxide, temperature and relative humidity. Int J Epidemiol 1982; 11:164–9.
34. Lesourd BM, Corriel RN, McBryde JL, Kniker WT. Cell mediated immunity in school children assessed by multitest CMI skin testing II. Epidemiologic factors affecting immune responsiveness. Ann Allergy; 1985; 54:446–52.
35. Varekamp H, Voorhorst R. De invloed van klimaat en behuizing op patienten met asthma bronchiale en rhinitis vasomotoria. Ned Tijdschr Geneeskd 1961; 105:2022–8.
36. Leupen MJ. Discussion. In: Fanger PO, Valbjørn O, eds. Copenhagen: Indoor Climate Danish Building Research Institute, 1979:124–5.
37. Ferris BG Jr. Project Coordinator. Epidemiology Standardization Project. Am Rev Respir Dis 1978; 118(Part 2:1–120).
38. Ware JH, Dockery DW, Spiro A III, Speizer FE, Ferris BG Jr. Passive smoking, gas cooking, and respiratory health of children living in six cities. Am Rev Respir Dis 1984; 129:366–74.
39. Tobin RS, Baranowski E, Gilman AP, Kuiper-Goodman T, Miller JD, Giddings M. Significance of fungi in indoor air: report from a working group. Can J Public Health 1987; 78:S1+S32.
40. Dockery DW, Berkey CS, Ware JH, Speizer FE, Ferris BG Jr. Distribution of FVC and FEV_1 in children 6 to 11 years of age. Am Rev Respir Dis 1983; 128:405–12.
41. Spengler JD, Ware J, Speizer F, *et al.* Harvard's indoor air quality respiratory health study. Proceedings of the 4th International Conference on Indoor Air Quality and Climate, Institute for Water, Soil and Air Hygiene, Berlin, GDR, 1987; 2:218–23.
42. SAS Institute Inc. SAS User's Guide: Statistics. Version 5. Cary, N.C.: SAS Institute, 1985:956.
43. Hallett JS, Jacobs RL. Recurrent acute bronchitis: the association with undiagnosed bronchial asthma (abstract). J Allergy Clin Immunol 1983; 71:97.
44. Strachan DP. Damp housing and childhood asthma: validation of reporting of symptoms. Br Med J 1988; 297:1223–6.
45. Dockery DW, Spengler JD, Speizer FE, Ferris BG Jr, Ware JH, Brunekreef B. Associations of health status with indicators of indoor air pollution from an epidemiologic study in six U.S. cities. Proceedings of the 4th International Conference on Indoor Air Quality and Climate, Berlin, GDR. 1987; 2:203–7.
46. Martin CJ, Platt SD, Hunt SM. Housing conditions and ill health. Br Med J 1987; 294:1125–7.